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A class of mathematical models of the irreversible lengthening of plant cells: An analysis of its generic properties by singular perturbation theory[1]

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Abstract: A system of nonlinear differential equations is posed to analyze the irreversible elongation (axial growth) of plant cells. The volume of the turgescent cell and its volume in the plasmolytic point are introduced as state variables. In the autonomous case, some of the qualitative properties of the orbits in phase plane, which have a greater interest from the perspective of growth, are determined. Two types of phase portraits are found. The transition (bifurcation) from one to the other occurs when the osmotic pressure between the vacuole and the cellular exterior (bifurcation parameter) crosses a critical value. For the geometry of orbits corresponding to the growth process itself, the general validity of an asymptotic property whose consequences have physiological interest is demonstrated. This asymptotic property deduced in the present work, was introduced as a hypothesis by Lockhart, in 1965. The hypothesis of Lockhart for a steady state growth process is justified here, and even more, a significant extension of this assumption is justified from a physical-mathematical point of view by analyzing the kinetic equations through the theory of singular perturbations, including the case in which extensibility, osmotic pressure, water permeability, critical turgor pressure, and the parameters of elasticity of the wall vary during growth. Two time scales are introduced, the plasmolysis scale (fast) and the growth scale (slow). Two different asymptotic solutions are constructed that are subsequently coupled to obtain a global solution: one of the solutions describes the relaxation of the state variables from their initial conditions, on the (fast) scale of plasmolysis, and the other solution describes its subsequent evolution, in the slow growth scale. This last solution is the one obtained if the Lockhart hypothesis is accepted. The method allows us to establish bounds to the error involved in using this hypothesis. In the discussion, some comments about the strength and weakness of mathematical modeling are given.

Key words: family of mathematical models, axial diffuse plant cell growth, Lockhart model, mechanical models of primary cell wall, phase portraits, bifurcation theory, singular perturbation theory, Liapunov-Perron characteristic numbers, multiple time scales, slow manifolds.

(1)-Introduction

In turgid plant cells, during the growth phase, there is an irreversible increase in the volume of the chamber limited by the primary cell wall, along with an increase in the cell surface area.

[1] This is the English translation of the 1984 paper, originally exposed in Spanish: "Una clase de modelos matemáticos que describen el alargamiento irreversible de células vegetales: análisis de sus propiedades genéricas mediante la teoría de perturbaciones singulares". A closely research article that also addresses the diffuse growth of the primary wall of plant cells was recently published, in Spanish, by the author: R. Suárez-Antola, "Un modelo matemático del crecimiento difuso de la pared primaria de células vegetales (A mathematical model of the diffuse growth of the primary wall of plant cells)", Rev. SCP, **23** (1): 13-34, 2018. This journal article stresses physiological and biophysical points of view, instead of the applied mathematics point of view adopted in the present conference paper. The 2018 research article describes the construction of a new mathematical model intended to study both the diffuse axial and radial growth of the primary wall of plant cells. Analytical formulae are obtained for Erickson's anisotropy quotient of growth and for the parameters of the Ortega´s augmented equation (a generalization of Lockhart's irreversible elongation equation), as a function of the parameters of the model. A non-linear constitutive relationship is introduced to describe axial growth. So, in the afore mentioned article, the physiological and biophysical contents of the present conference paper related to the diffuse growth of the primary wall of plant cells are updated, expanded, and significantly generalized. However, the main results of the analysis done in the 1984 paper from the standpoint of biomathematics, and here translated to English, remained unpublished.

The growth of the primary wall can be analyzed from a biophysical perspective, as a mechanical-hydraulic process, dependent on both the mechanical properties of the cell wall and the osmotic properties of the cytoplasm.
An operative difference in osmotic $\Delta\pi$ and hydraulic pressure (turgor pressure) ΔP can be defined between the vacuole and the cell environment.

During cell wall growth the increase in volume in general occurs without an increase in mechanical stresses inside the cell wall. If the stiffness of the wall did not decrease during the expansion of the wall, the turgor pressure would increase and with it the mechanical stresses inside the wall would increase.
The imbalance between osmotic pressure and turgor pressure, and therefore growth, can be sustained over time.
The biochemical mechanisms of these changes in the mechanical properties of the wall and their regulation are not fully understood at present but are being intensively investigated by applying the tools of contemporary molecular biology and plant physiology. However, in this work the growth of the cell wall is considered from the **mechanical-hydraulic** point of view. The effects due to biochemical mechanisms appear implicitly in some of the phenomenological parameters that are introduced during the construction of the generic mathematical models.

The expansion of the cell wall may be highly localized (growth at the ends) or it may be produced distributed (diffuse growth). The generic mathematical models considered here describe diffuse growth.
The degree of anisotropy that diffuse wall expansion can present depends on how the new cellulose fibers deposited on its inner face are distributed. If the angular distribution is uniform, the expansion is going to be isotropic. If the angular distribution is not uniform, and most of the fibers tend to be parallel, the expansion will be anisotropic. The elongation will be greater in the direction perpendicular to the predominant direction in the fibers of the inner face of the wall.
As the formation of new wall continues while the wall expands, each layer of fibers stretches and thins. Its fibers are reoriented in the direction of growth, so that the outermost (oldest) layers have fibers that form smaller angles with respect to the direction of growth. This mode of growth was raised by Roelofsen and Houwink in 1953 as the so-called hypothesis of the multiple network (Mühlethaler, 1967; Noble, 1974; Erickson, 1980).

A first mathematical model of irreversible plant cell elongation, published in 1965, is the one constructed and analyzed by Lockhart. This author begins with two coupled kinetic equations, one for osmotic water absorption and another for irreversible expansion of the cell wall.
The rate of variation of the volume V of the turgid cell can be related to the volume flow density (mostly a water flow) through a permeability barrier established between the extracellular space and the tonoplast and with the area S of the cell wall through which the flow occurs. If t represents the instant of time , K_H is the hydraulic permeability, $\Delta\pi$ is the osmotic pressure difference, ΔP is the turgor pressure, the equation for osmotic balance can be written as follows:

$$\frac{dV}{dt} = S\, K_H\,\left(\Delta\pi - \Delta P\right) \qquad [1\ a]$$

The difference in osmotic pressure $\Delta\pi$ between the vacuolar fluid and the extracellular space, results from the contribution of numerous solutes. The degree of impermeability of the barrier with respect to solutes can be quantified using Stavermann's reflection coefficients (Katchalsky and Curran, 1967).
The hydraulic permeability K_H corresponds to a barrier composed of a series arrangement of cell wall, plasma membrane, cytoplasm and tonoplast.
Both for the establishment of the osmotic pressure difference and for the determination of the value of hydraulic permeability the cell wall is not the most important component (Harris, 1960; Dainty, 1963). But it is when considering the elastic response of the turgid cell (Green, Erickson and Buggy, 1971; Noble, 1974; Wainwright et al, 1976).

Equation [1 a] can be rewritten:

$$\frac{1}{V}\,\frac{dV}{dt} = \mu\, K_H\,\left(\Delta\pi - \Delta P\right) \qquad [1\ b]$$

In [1 b], by definition: $$\mu = \frac{S}{V} \qquad [2]$$

The equation for the irreversible expansion of the cell wall can be written as follows, being V_0 the cell volume in incipient plasmolysis:

$$\frac{1}{V_0}\frac{dV_0}{dt} = \phi_0 \cdot (\Delta P - \Delta P_c) \cdot H_{\Delta P_c}(\Delta P) \qquad [3]$$

In equation [3], ϕ_0 is a measure of the extensibility of the wall, ΔP_c is a critical value of ΔP below which the cell wall only deforms reversibly and above which it can deform irreversibly, and $H_{\Delta P_c}(\Delta P)$ is Heavside´s unit step function. [2]

Lockhart represents the plant cell by a cylinder of variable length and constant radius. It uses as state variables the length of the turgid cell L and the length L_0 of the cell in incipient plasmolysis. It assumes elastic balance and a linear elastoplastic wall (Lockhart, 1965).
In this case: $V = \pi R^2 L$ and $V_0 = \pi R^2 L_0$ **being, by hypothesis, R constant**. Besides, Lockhart assumes that the exchange area is $S = 2\pi R L$.
The kinetic equations [1 b] and [3] of the mathematical model can be reformulated:

$$\frac{1}{L}\frac{dL}{dt} = \frac{2}{R} K_H (\Delta\pi - \Delta P) \qquad [4\ a]$$

$$\frac{1}{L_0}\frac{dL_0}{dt} = \phi(\Delta P - \Delta P_c) H_{\Delta P_c}(\Delta P) \qquad [4\ b]$$

In [4 b], by definition: $\phi = \frac{2}{R}\phi_0$

For the case in which the osmotic pressure difference exceeds the critical turgor pressure ΔP_c, Lockhart introduced **an additional hypothesis**:

$$\frac{1}{L}\frac{dL}{dt} = \frac{1}{L_0}\frac{dL_0}{dt} = r_g \qquad [5]$$

From equations [4 a], [4 b] and [5], respectively, a formula expressing the cell elongation rate r_g and a formula for water potential M are obtained, depending on the osmotic pressure, the extensibility of the wall, water permeability and cell radius:

$$r_g = \frac{(2K_H/R)}{(\varphi + 2K_H/R)} \varphi (\Delta\pi - \Delta P_c) \qquad [6]$$

$$M = -\frac{\varphi}{\varphi + (2K_H/R)} (\Delta\pi - \Delta P_c) \qquad [7]$$

If the extensibility, osmotic pressure, water permeability and critical turgor pressure remain constant, according to [6] the cell elongation rate r_g remains constant. Then from [5] an exponential elongation of the primary wall is obtained:

$$L_0(t) = L_0(0) \exp[r_g t] \qquad [8]$$

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[2] By definition $H_{\Delta P_c}(\Delta P)$ is equal to 1 if $\Delta P \geq \Delta P_c$ and it is equal to 0 when $\Delta P < \Delta P_c$

In addition, Lockhart studied other cases: decreasing extensibility in the growth time scale, osmotic pressure dependent on cell volume in the plasmolysis time scale, etc. When some parameters are adjusted, the deduced formulas predict growth curves compatible with those observed experimentally (Lockhart, 1965).

The quotient between the thickness h of the wall and its radius R_0 in incipient plasmolysis is in general more than one order of magnitude less than 1. [3] Then the turgor pressure can be related with the average axial stress σ_z and the wall radius applying the elementary theory of cylindrical shells (Timoshenko and Goodier, 1951):

$$\Delta P = \frac{2h}{R}\sigma_z \qquad [9]$$

If the wall material behaves as an equivalent homogeneous and linear elastic material, the relation between the axial stress σ_z and the axial strain $\varepsilon_z = \frac{L-L_0}{L_0}$ will be given by Hook law, being E the Young's modulus:

$$\sigma_z = E\left(\frac{L-L_0}{L_0}\right) \qquad [10]$$

From equations [9] and [10], we substitute $\Delta P = \frac{2h}{R}E\left(\frac{L-L_0}{L_0}\right)$ in equations [4 a] and [4 b]:

$$\frac{1}{L}\frac{dL}{dt} = \frac{2}{R}K_H\left(\Delta\pi - \frac{2h}{R}E\left(\frac{L-L_0}{L_0}\right)\right) \qquad [11\ a]$$

$$\frac{1}{L_0}\frac{dL_0}{dt} = \phi\left(\frac{2h}{R}E\left(\frac{L-L_0}{L_0}\right) - \Delta P_c\right)H_{\Delta P_c}\left(\Delta P\right) \qquad [11\ b]$$

From equation [9] the critical turgor pressure ΔP_c can be related with an axial tensile yield stress $\sigma_{z,c}$ of the wall material:

$$\Delta P_c = \frac{2h}{R}\sigma_{z,c} \qquad [12]$$

This result allows us to re-write [11 b]:

$$\frac{1}{L_0}\frac{dL_0}{dt} = \phi\frac{2h}{R}\left(E\left(\frac{L-L_0}{L_0}\right) - \sigma_{z,c}\right)H_{\sigma_{z,c}}\left(\sigma_z\right) \qquad [13]$$

Now $H_{\sigma_{z,c}}\left(\sigma_z\right)$ is Heavside's unit step function in terms of axial stresses.

Later, Erickson constructed and analyzed a diffuse growth model of the primary cell wall for a cylindrical cell whose wall expands both axially and radially (Erickson, 1980).

It assumed an exponential axial growth as represented in [8] and introduced a growth anisotropy ratio to connect the axial and radial expansions:

$$\frac{d\,Ln\left(L_0(t)\right)}{d\,Ln\left(R_0(t)\right)} = k \qquad [14]$$

Then the radius in incipient plasmolysis verifies (Erickson, 1980):

$$R_0(t) = R_0(0)\exp\left[\frac{r_g}{k}t\right] \qquad [15]$$

If it is assumed that the angular distribution of the fibers that are deposited on the inner face of the wall is uniform, the wall expansion will be fundamentally isotropic. In this case the cell can be represented by a sphere and the variations in cell volume, both in the scale of plasmolysis and in the scale of growth, can be described by a mathematical model

[3] For Nitella or Chara cells, a radius of 0.5 mm and a primary wall thickness of 5 μm can be estimated, so that $\frac{R}{h} \approx 100$ (Frey-Wyssling, 1952; Wainwright et al, 1976; Erickson, 1980)

analogous to Lockhart's. On the scale of plasmolysis, the radius R_0 of the cell in incipient plasmolysis can be considered constant.
With this we finalize our review of the equations Lockhart's mathematical model and now we can establish the objectives of the paper.
The main purpose is to show that the additional hypothesis $\frac{1}{L}\frac{dL}{dt}=\frac{1}{L_0}\frac{dL_0}{dt}$ introduced by Lockhart can be justified from the physical-mathematical point of view, by analyzing the kinetic equations employing the theory of singular perturbations.
We show here that Lockhart's hypothesis can be justified even in a more general case in which the extensibility, osmotic pressure, water permeability, critical turgor pressure and elasticity parameters of the wall vary during growth, that is, Lockhart's hypothesis can be justified for a whole class of mathematical models.
To enable the application of singular perturbation theory, two widely separated time scale, the scale of plasmolysis (fast) and the scale of the plant cell growth (slow) are introduced.
Two different asymptotic solutions are constructed that are later combined to obtain a global solution.
One of the asymptotic solutions describes the relaxation of the state variables from their initial conditions, on the fast scale of plasmolysis, towards a so-called slow manifold.
The other asymptotic solution describes their later evolution in the growth scale, on the slow manifold, considered in the sense of singular perturbation theory [4]. This last solution is the one obtained if the hypothesis $\frac{1}{L}\frac{dL}{dt}=\frac{1}{L_0}\frac{dL_0}{dt}$ is accepted. The method allows us to introduce bounds to the error involved in using Lockhart's hypothesis.

(2)-A class of mathematical models of the irreversible lengthening of plant cells

On the scale of growth some of the parameters that were introduced by Lockhart in the mathematical model may undergo non-negligible modifications, in time and sometimes also in space (non-spatial homogeneity).
Besides, the hypothesis of linear elastic behavior of the cell wall material could be in some cases too restrictive.
Although spatial heterogeneity will not be studied in this paper, the effects of the time variation of some parameters and the nonlinearity of the elastic stress-strain constitutive relation will be considered, as well as the nonlinearity of both the elastic response of the wall in the fast time scale of plasmolysis and its plastic response with synthesis of new wall material in the time scale of growth.

The volume of the turgescent cell V and its volume V_0 in the point of incipient plasmolysis are introduced as state variables. Instead of equation [3] for the irreversible expansion of the cell wall, we suppose that the plastic mechanical-chemical response is given by:

$$\frac{1}{V_0}\frac{dV_0}{dt}=h\left(t,\Delta P\right) \qquad [16]$$

[4] A manifold is a topological space that locally, near each of its points, resembles a Euclidean space: each point of a n-dimensional manifold has a neighborhood that is homeomorphic to the Euclidean space of dimension n. An invariant manifold of a dynamic system is a manifold made with orbits of the dynamic system (Hirsch and Smale, 1974). "Slow manifold" can be used with two different meanings. Some people refer to slow manifolds in the framework of the so called central manifold theory (Haken, 1983): in this case a slow manifold is always a central one and as consequence is an invariant manifold. Other people understand slow manifold in the frame of singular perturbation theory (Tihonov, Vasileva and Volosov, 1970) as will be done here. Taken in this last sense, a slow manifold is not invariant.

The plastic response function $h(t,\Delta P)$ is non negative, smooth function of t and ΔP that is zero if $\Delta P \le \Delta P_c$ and verifies $\frac{\partial}{\partial \Delta P} h(t,\Delta P) > 0$ when both $\Delta P_c < \Delta P$ and $h(t,\Delta P) > 0$. Here ΔP_c is related with the yield stress trough equation [12] of the introduction. The explicit time dependence allows us to consider the physicochemical modifications of the cell wall during the growth process.

The nonlinearity of the elastic response will be represented here by a smooth relation between cubic dilatation $\Theta = \frac{V}{V_0} - 1$ and hydraulic pressure:

$$\Delta P = g(t,\Theta) \qquad [17]$$

Again, the explicit time dependence allows us to consider the physicochemical modifications of the cell wall, including the possible redistribution of elastic microfibers in the wall amorphous matrix.

For every time instant and when $\Theta \le 0$ we suppose that $g(t,\Theta) = 0$.

When $\Theta > 0$, we assume $\frac{\partial}{\partial \Delta P} g(t,\Delta P) > 0$ also for every time instant.

The corresponding generalizations of equations [1 b] and [3] are:

$$\frac{1}{V}\frac{dV}{dt} = \mu K_H \left(\Delta\pi - g(t,\Theta)\right) = a(t,\Theta) \qquad [18\ a]$$

$$\frac{1}{V_0}\frac{dV_0}{dt} = h\left(t, g(t,\Theta)\right) = b(t,\Theta) \qquad [18\ b]$$

$$\frac{V}{V_0} = (1+\Theta) \qquad [18\ c]$$

From [18 a], [18 b] and [18 c] a single equation in terms of the cubic dilatation results:

$$\frac{d\Theta}{dt} = (1+\Theta)\, c(t,\Theta) \qquad [19]$$

In [19], by definition: $$c(t,\Theta) = a(t,\Theta) - b(t,\Theta) = \mu K_H \left(\Delta\pi - g(t,\Theta)\right) - h\left(t, g(t,\Theta)\right) \qquad [20]$$

From equation [19] the cubic dilatation $\Theta(t) = \frac{V(t)}{V_0(t)} - 1$ can be calculated, considering its initial value $\Theta(0) = \frac{V(0)}{V_0(0)} - 1$. If we know $\Theta(t)$, then the volume of the turgid cell and its volume at incipient plasmolysis can be determined from [18 a] and [18 b], respectively, considering it initial values:

$$V(t) = V(0)\exp\left[\int_0^t a\left(s,\Theta(s)\right) ds\right] \qquad [21\ a]$$

$$V_0(t) = V_0(0)\exp\left[\int_0^t b\left(s,\Theta(s)\right) ds\right] \qquad [21\ b]$$

To finish defining the class of generic mathematical models that describe the irreversible lengthening of plant cells we add the assumption that $\mu = \frac{S}{V} = \frac{2\pi R L}{\pi R^2 L} = \frac{2}{R}$ is fixed because the cell wall radius R remains constant, by hypothesis, during the growth process.

In what follows, **the difference in osmotic pressure** $\Delta\pi$ between the vacuolar fluid and the extracellular space **will be considered as always positive**. If $\Delta\pi$ is negative, the protoplasm contracts and will tend to detach from the interior face of the cell wall shell.

(2.1)-Fast volume changes during plasmolysis

In the fast scale of plasmolysis, the mechanisms that regulate osmotic pressure in vacuole could be too slow to compensate the fast change in cell volume. If their regulatory effect can be neglected, the difference in osmotic pressure between the vacuole and the cell environment can be approximated by the function $\Delta\pi = \frac{\left(V_0 - V_\odot\right)}{V - V_\odot}\pi_{i,0} - \pi_e$

In this expression π_e is the osmotic pressure of the environment and $\frac{\left(V_0 - V_\odot\right)}{V - V_\odot}\pi_{i,0} = \pi_i\left(V\right)$ is the osmotic pressure in the vacuole. The parameter $V_\odot$ can be interpreted as a limit volume at which V approaches when the amount of intra-vacuolar water decreases.

Now V_0 remains constant so $\frac{V_\odot}{V_0}$ is constant and the difference in osmotic pressure can be written this way:

$$\Delta\pi\left(\Theta\right) = \pi_i\left(\Theta\right) - \pi_e = \frac{\left(1 - \frac{V_\odot}{V_0}\right)}{\left(1+\Theta\right) - \frac{V_\odot}{V_0}}\pi_{i,0} - \pi_e \qquad [22]$$

In equation [20] the plastic response function that appears in the left-hand member of [18 b] can be neglected, the elastic characteristic is stationary and consequently, in equation [19] we have:

$$c\left(\Theta\right) = a\left(\Theta\right) = \mu K_H\left(\Delta\pi\left(\Theta\right) - g\left(\Theta\right)\right) = \mu K_H\left(\frac{\left(1 - \frac{V_\odot}{V_0}\right)}{\Theta + \left(1 - \frac{V_\odot}{V_0}\right)}\pi_{i,0} - \left(\pi_e + g\left(\Theta\right)\right)\right) \qquad [23]$$

Considering [23] we see that equation [19] can be simplified:

$$\frac{d\,\Theta}{d\,t} = \left(1+\Theta\right)\,a\left(\Theta\right) = f\left(\Theta\right) \qquad [24]$$

We know that $\Delta\pi\left(\Theta\right)$ is positive and decreases monotonically while $g\left(\Theta\right)$ increases monotonically from zero when Θ increase so we have a single positive root Θ_∞ for the equation $a\left(\Theta\right) = 0$.

Equation [24] could be solved giving time as a function of the cubic dilatation, either analytically (by separation of variables for suitable $f\left(\Theta\right)$) or numerically. However, let now us consider the local stability of the rest point Θ_∞.

Introducing $\xi = \Theta - \Theta_\infty$ in [24]:

$$\frac{d\,\xi}{d\,t} = f\left(\Theta_\infty + \xi\right) = \left[\frac{d}{d\,\Theta}f\left(\Theta_\infty\right)\right]\xi + o\left(\xi\right) = -\frac{1}{\tau_p}\xi + o\left(\xi\right) \qquad [25]$$

As usual $o\left(\xi\right)$ is such that $\frac{o\left(\xi\right)}{\xi}$ tends to zero with ξ. In [25] appears a characteristic measure of time, the time of relaxation of Θ towards Θ_∞ during a process of uncompensated (by osmotic regulatory mechanisms) plasmolysis:

$$\tau_p = -\frac{1}{\left(1+\Theta_\infty\right)\frac{d}{d\Theta}a\left(\Theta_\infty\right)} \qquad [26]$$

From [23] it is possible to determine $\frac{d}{d\Theta}a\left(\Theta_\infty\right) = \mu K_H \left(\frac{d}{d\Theta}\Delta\pi\left(\Theta\right) - \frac{d}{d\Theta}g\left(\Theta\right)\right)$

In the framework of the fast volume changes during plasmolysis $\frac{d}{d\Theta}\Delta\pi\left(\Theta\right)$ is always negative, and in the framework of the mathematical models being considered $\frac{d}{d\Theta}g\left(\Theta\right)$ is always positive. So $\frac{d}{d\Theta}a\left(\Theta_\infty\right)$ is negative, and consequently τ_p positive. Further details can be found elsewhere (Suárez-Antola, 1985).

(2.2)- The nonlinear osmometer: the Liapunov-Perron characteristic numbers and Lockhart hypothesis

From [18 a], [18 b] and [20] we obtain:

$$\frac{1}{V}\frac{dV}{dt} - \frac{1}{V_0}\frac{dV_0}{dt} = c\left(t,\Theta\right) \qquad [27]$$

Lockhart hypothesis (equation [5] in the Introduction to this paper) would be now, in terms of volumes, $\frac{1}{V}\frac{dV}{dt} = \frac{1}{V_0}\frac{dV_0}{dt}$ or (equivalently) $c\left(t,\Theta\right) = 0$ in equation [27].

This is not the case during the fast volume changes that occur in plasmolysis processes, as was analyzed in the previous subsection (2.1) “Fast volume changes during plasmolysis”, with $c\left(t,\Theta\right) = c\left(\Theta\right) = a\left(\Theta\right)$ given by [23].

However, Lockhart hypothesis could be a good approximation in the slower scale of growth.

(2.2.1)- The autonomous case.

To study if this is so, let us consider first the autonomous case, with $\Delta\pi$ constant and time independent elastic $g\left(\Theta\right)$ and plastic $h\left(g\left(\Theta\right)\right)$ characteristic functions:

$$\frac{d\Theta}{dt} = \left(1+\Theta\right)c\left(\Theta\right) \qquad [28\ a] \qquad\qquad c\left(\Theta\right) = a\left(\Theta\right) - b\left(\Theta\right) \qquad [28\ b]$$

$$a\left(\Theta\right) = \mu K_H \left(\Delta\pi - g\left(\Theta\right)\right) \qquad [28\ c] \qquad\qquad b\left(\Theta\right) = h\left(g\left(\Theta\right)\right) \qquad [28\ d]$$

$$\frac{1}{V}\frac{dV}{dt} = a\left(\Theta\right) \qquad [29\ a] \qquad\qquad \frac{1}{V_0}\frac{dV_0}{dt} = b\left(\Theta\right) \qquad [29\ b]$$

Equation [28 a], in addition to the unphysical rest point $\Theta = -1$, has only other rest point Θ_∞. It verifies $c\left(\Theta_\infty\right) = 0$.

Indeed, keeping in mind the assumed properties for the functions $g\left(\Theta\right)$ and $h\left(\Delta P\right)$, from [28 c] and [28 d] it follows:

(a) When Θ increases from zero, $a\left(\Theta\right)$ decreases monotonically and continuously from $\mu K_H \Delta\pi > 0$ up to negative values, crossing zero in Θ_* such that $g\left(\Theta_*\right) = \Delta\pi$. The slope $\frac{d}{d\Theta}a\left(\Theta_*\right)$ is always negative.

(b) When increases from zero, $b\left(\Theta\right)$ is zero until $g\left(\Theta\right) \le \Delta P_c$ and after that it increases monotonically.

As consequence, the graphs of these functions cross each other when $\Theta = \Theta_\infty$, the only root of $c\left(\Theta\right) = 0$. See Figure 1.

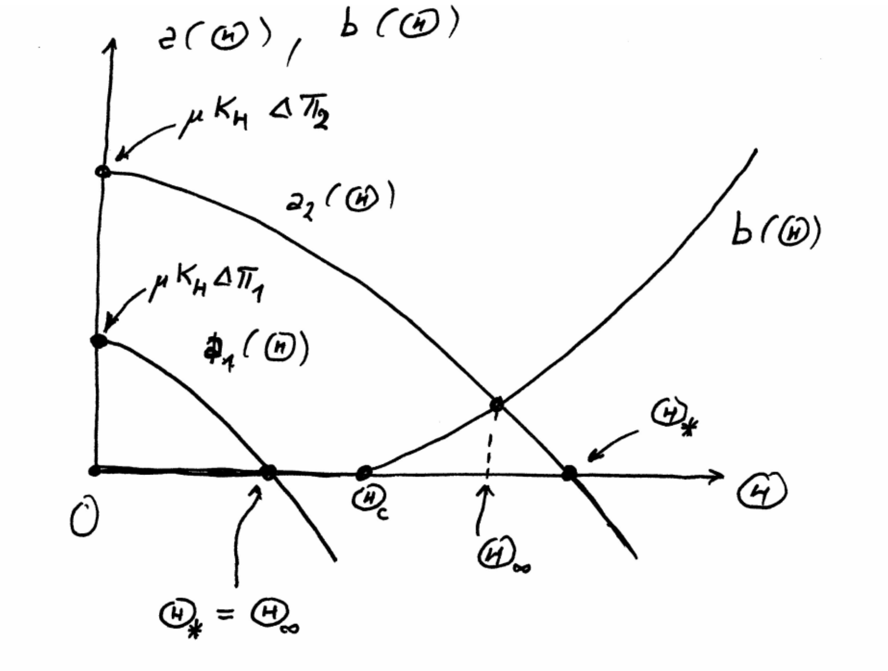

Figure 1. Qualitative graphs of the functions $a(\Theta)$ and $b(\Theta)$, where $a_1(\Theta)$ corresponds to $\Delta\pi < \Delta P_c$ and $a_2(\Theta)$ corresponds to $\Delta\pi > \Delta P_c$ If $\Delta\pi < \Delta P_c$ it follows $\Theta_\infty = \Theta_* < \Theta_c$ If $\Delta\pi > \Delta P_c$ it follows $\Theta_c < \Theta_\infty < \Theta_*$ Besides $a(\Theta_\infty) = b(\Theta_\infty)$ and as consequence $c(\Theta_\infty) = 0$

Please note that from [27] it follows $\left(\frac{1}{V}\frac{dV}{dt}\right)_{\Theta=\Theta_\infty} - \left(\frac{1}{V_0}\frac{dV_0}{dt}\right)_{\Theta=\Theta_\infty} = c(\Theta_\infty) = 0$, consequently, when the dynamics is described by an autonomous system, Lockhart hypothesis is always verified in the steady state of the cell, corresponding to $\Theta = \Theta_\infty$.

Introducing the turgor pressure $\Delta P = g(\Theta)$ it is possible to recast $c(\Theta)$ as a function of this pressure:

$c(\Theta) = \mu K_H \left(\Delta\pi - \left[\Delta P + \frac{1}{\mu K_H} h(\Delta P)\right]\right)$ Keeping in mind that $h(\Delta P) \geq 0$, if $c(\Theta_\infty) = 0$ and $\Delta P_\infty = g(\Theta_\infty)$ we have $\Delta P_\infty \leq \Delta\pi$.

If $\Delta\pi < \Delta P_c$, $h(\Delta P_\infty) = 0$ and $\Delta P_\infty = \Delta\pi$. In this case the cell wall doesn't growth (V_0 remains constant) in the corresponding equilibrium $\Theta = \Theta_\infty = \Theta_* < \Theta_c$:

$$\left(\frac{1}{V_0}\frac{dV_0}{dt}\right)_\infty = h(\Delta P_\infty) = b(\Theta_\infty) = 0$$

If $\Delta\pi > \Delta P_c$, as consequence of the monotonic behavior of $\Delta P + \frac{1}{\mu K_H} h(\Delta P)$ we have $\Delta P_\infty < \Delta\pi$ and $h(\Delta P_\infty) > 0$. So, in this case the cell wall grows in steady state (V_0 increases). Now: $0 < \Theta_c < \Theta_\infty < \Theta_*$

The transient behavior of the cell wall follows from the equation [28 a] (for $\Theta \geq 0$) jointly with [29 a] and [29 b].

Equation [28 a] can be written $\frac{d\,\Theta}{d\,t} = f(\Theta)$ with $f(\Theta) = (1+\Theta)\,c(\Theta)$ being negative if $0 \le \Theta < \Theta_\infty$ and positive if $\Theta_\infty < \Theta$. So the rest point Θ_∞ is globally and asymptotically stable (Coddington and Levinson, 1955; Han, 1967; Saaty and Bram, 1981): for any initial condition $0 \le \Theta(0)$, for every possible trajectory $\Theta(t;\Theta(0))$ we have $\lim_{t\to\infty} \Theta(t;\Theta(0)) = \Theta_\infty$

To get more detail about this limit behavior, let us calculate the Liapunov-Perron numbers for these trajectories.

In order to do this, we define $\xi = \Theta - \Theta_\infty$ and keeping in mind that $f(\Theta_\infty) = 0$ and the function $f(\Theta)$ is smooth, let us work with the following Taylor development:

$$f(\Theta) = f(\Theta_\infty + \xi) = \frac{d}{d\,\Theta} f(\Theta_\infty)\xi + \frac{1}{2}\frac{d^2}{d\,\Theta^2} f(\Theta_\infty)\xi^2 + \frac{1}{6}\frac{d^3}{d\,\Theta^3} f(\Theta_\infty)\xi^3 + \cdots \qquad [30]$$

The coefficient of the first $\frac{d}{d\,\Theta} f(\Theta_\infty) = (1+\Theta_\infty)\frac{d}{d\,\Theta} c(\Theta_\infty)$ is always negative.

Define the characteristic time:

$$\tau_\Theta = -\frac{1}{(1+\Theta_\infty)\frac{d}{d\,\Theta} c(\Theta_\infty)} \qquad [31]$$

Then [30] can be recast as follows

$$f(\Theta) = -\frac{1}{\tau_\Theta}\xi\left(1+\varphi(\xi)\right) \qquad [32]$$

The properties of the function $f(\Theta)$ imply that $1+\varphi(\xi) > 0$ if $\xi \ne 0$, and $\varphi(\xi) = O(\xi)$. Equation [28 a], in terms of the new variable ξ can be written:

$$\frac{d\,\xi}{d\,t} = -\frac{1}{\tau_\Theta}\xi\left(1+\varphi(\xi)\right) \qquad [33]$$

Let us consider any solution of [33] with $\xi(0) \ne 0$: $\quad \xi(t;\xi(0)) = \Theta(t;\Theta(0)) - \Theta_\infty$

From [33] we obtain:

$$\frac{1}{t}\log_e \frac{|\xi(t;\xi(0))|}{|\xi(0)|} = -\frac{1}{\tau_\Theta}\left(1 + \frac{1}{t}\int_0^t \varphi(\xi(s;\xi(0)))\,d\,s\right) \qquad [34]$$

By definition, the Liapunov-Perron number of a trajectory is $\lambda = \limsup_{t\to\infty} \frac{1}{t}\log_e |\xi(t;\xi(0))|$ (Liapunov, 1947; Perron, 1930; Malkin, 1952) or equivalently $\lambda = \limsup_{t\to+\infty} \frac{1}{t}\log_e \frac{|\xi(t;\xi(0))|}{|\xi(0)|}$

The Liapunov-Perron number of the trajectory $\xi(t;\xi(0))$ can be calculated from equation [34], considering that $\lim_{t\to+\infty}\frac{1}{t}\int_0^t \varphi(\xi(s;\xi(0)))\,d\,s = 0$:

$$\lambda = \limsup_{t\to+\infty}\frac{1}{t}\log_e \frac{|\xi(t;\xi(0))|}{|\xi(0)|} = \lim_{t\to+\infty}\frac{1}{t}\log_e \frac{|\xi(t;\xi(0))|}{|\xi(0)|} = -\frac{1}{\tau_\Theta} \qquad [35]$$

We have **the same Liapunov-Perron number** for all possible trajectories that approach to the rest point $\xi = 0$.

Please note that as the rest point $\xi = 0$ is globally and asymptotically stable, the function $\varphi(\xi(t;\xi(0)))$ tends to zero when $t \to +\infty$ and then it is possible to derive $\lim_{t\to+\infty}\frac{1}{t}\int_0^t \varphi(\xi(s;\xi(0)))\,d\,s = 0$ [5]

From [35] it follows that asymptotically, all trajectories $\Theta(t;\Theta(0))$ approaches exponentially to Θ_∞ with the same characteristic time τ_Θ

[5] Se the lemma in Appendix (5.1)

So, asymptotically $c(\Theta)$ approaches to zero. In this autonomous case, from [27]: $\frac{1}{V}\frac{dV}{dt}-\frac{1}{V_0}\frac{dV_0}{dt}=c(\Theta)$

So, the hypothesis introduced by Lockhart is verified asymptotically when $t\to+\infty$.

An estimation of the duration of the transient leading to the equality of the relative rates of variation of the volume of the turgid cell and the cell in incipient plasmolysis, can be obtained from the characteristic time τ_Θ

When Θ approaches to Θ_∞, from [29 a] we find that $\frac{1}{V}\frac{dV}{dt}$ approaches to $a(\Theta_\infty)$, and from [29 b] we find that $\frac{1}{V_0}\frac{dV_0}{dt}$ approaches to $b(\Theta_\infty)=a(\Theta_\infty)=\lambda_\infty$. Then, asymptotically when $t\to+\infty$:

$$V(t)\approx\bar{V}(0)\exp[\lambda_\infty t] \qquad [36\ a] \qquad\qquad V_0(t)\approx\bar{V}_0(0)\exp[\lambda_\infty t] \qquad [36\ b]$$

Because $\frac{V(t)}{V_0(t)}=1+\Theta(t)$ and $\Theta(t)$ tends to Θ_∞ while $\frac{\exp[a(\Theta_\infty)t]}{\exp[b(\Theta_\infty)t]}=\exp[c(\Theta_\infty)t]=1$, the volumes $\bar{V}(0)$ and $\bar{V}_0(0)$ must be related by the formula:

$$\frac{\bar{V}(0)}{\bar{V}_0(0)}=1+\Theta_\infty \qquad [37]$$

From [21 a] and [21 b], the trajectories in the autonomous case are:

$$V(t;V(0))=V(0)\exp\left[\int_0^t a(\Theta(s))ds\right] \qquad [38\ a] \qquad\qquad V_0(t;V_0(0))=V_0(0)\exp\left[\int_0^t b(\Theta(s))ds\right] \qquad [38\ b]$$

Then: $$\frac{1}{t}\log_e\frac{V(t;V(0))}{V(0)}=\frac{1}{t}\int_0^t a(\Theta(s))ds \qquad\qquad \frac{1}{t}\log_e\frac{V_0(t;V_0(0))}{V_0(0)}=\frac{1}{t}\int_0^t b(\Theta(s))ds$$

From the limits $\lim_{t\to\infty}a(\Theta(t))=a(\Theta_\infty)$ $\lim_{t\to\infty}b(\Theta(t))=b(\Theta_\infty)=a(\Theta_\infty)$ and the definition of a Liapunov-Perron number for a trajectory, and from the lemma (5.1) in the Appendix, it follows that the Liapunov-Perron numbers for the turgid volume $V(t)$ and the volume at incipient plasmolysis $V_0(t)$ are both equal to λ_∞.

Let us consider now the orbits corresponding to the trajectories of the autonomous system of nonlinear differential equations with smooth right hand members $\frac{dV}{dt}=a(\Theta)V$, $\frac{dV_0}{dt}=b(\Theta)V_0$ (with $\Theta=\frac{V}{V_0}-1$) in the phase plane of coordinates V_0 and V. Please note that $b(\Theta)=h(\Delta P)=h[g(\Theta)]$ is always nonnegative while $a(\Theta)=\mu K_H(\Delta\pi-\Delta P)=\mu K_H(\Delta\pi-g(\Theta))$ changes it sign from positive to negative only once as Θ grows from zero.

A qualitative analysis of the portraits of the orbits in the phase plane can be done applying a well-known systematic procedure (Davis, 1962). After identifying the rest points, the regions where $\frac{dV}{dV_0}$ is either positive or negative and their boundary curves where $\frac{dV}{dV_0}$ is zero or ∞ are determined, as well as the regions where $\frac{d^2V}{dV_0^{\,2}}$ is either positive

or negative and the boundary curves where it is zero. So, the slope, concavity and inflection points of the orbits can be determined, and a qualitative portrait of the orbits in the phase plane can be constructed.
In the case of cell wall growth, only the first quadrant of the phase plane must be considered, and in this first quadrant only the region $\frac{V}{V_0}=(1+\Theta)\geq 1$ (or equivalently $\Theta \geq 0$) is of interest in the framework of the present mathematical model. The origin $V=V_0=0$ is excluded also from the set of possible states (V_0, V) of the system.

When $\Theta > \Theta_c, b(\Theta)>0$ and $\frac{dV}{dV_0}=\frac{a(\Theta)}{b(\Theta)}\frac{V}{V_0}=(1+\Theta)\frac{a(\Theta)}{b(\Theta)}$ $\quad \frac{d^2V}{dV_0^2}=\left(\frac{d}{d\Theta}(1+\Theta)\frac{a(\Theta)}{b(\Theta)}\right)\left(\frac{a(\Theta)}{b(\Theta)}-1\right)$

So the sign of the slope $\frac{dV}{dV_0}(\Theta)$ is equal to the sign of $a(\Theta)$: being Θ_* the only root of $a(\Theta)=0$, the slope $\frac{dV}{dV_0}$ is positive if $\Theta<\Theta_*$, negative if $\Theta>\Theta_*$ and zero when $\Theta=\Theta_*$

When $\Theta \leq \Theta_c$, $b(\Theta)=0$ so $\frac{dV}{dV_0}(\Theta)=\pm\infty$ (the slope is $+\infty$ if $\Theta<\Theta_\infty$ and $-\infty$ if $\Theta>\Theta_\infty$)

When $\Theta>\Theta_c$ ($b(\Theta)>0$) when Θ is near enough to Θ_c, $\frac{d}{d\Theta}(1+\Theta)\frac{a(\Theta)}{b(\Theta)}$ is positive while $\frac{a(\Theta)}{b(\Theta)}-1$ is negative.

As consequence, the sign of $\frac{d^2V}{dV_0^2}(\Theta)$ is negative when Θ is near enough to Θ_c. See Figure 2 a sketch of the phase portrait when $\Delta\pi<\Delta P_c$.

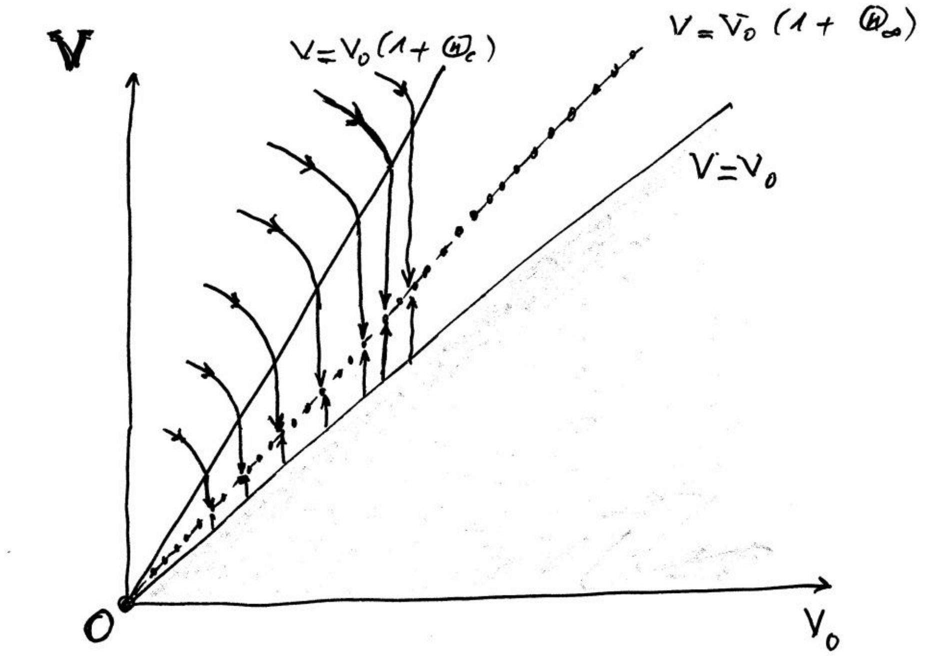

Figure 2. A qualitative sketch of the orbits when $\Delta\pi<\Delta P_c$

As shown in Figure 2, all the points of the half-straight line $\frac{V}{V_0} = 1 + \Theta_\infty \quad V_0 > 0$ are stable equilibrium points of the dynamics. Between $\frac{V}{V_0} = 1$ and $\frac{V}{V_0} = 1 + \Theta_c$ the segments of orbit are parallel to the axis of ordinates: V_0 remains constant and there is no plastic strain and no growth.

Above $\frac{V}{V_0} = 1 + \Theta_c$ the volume V_0 increases and V decreases: there is some irreversible deformation, and the turgid volume relaxes as shown.

Figure 3 shows a sketch of the orbits when $\Delta\pi > \Delta P_c$. In this case the half-straight line $\frac{V}{V_0} = 1 + \Theta_\infty \quad V_0 > 0$ is an orbit which attracts the other orbits. Asymptotically, both state variables (V_0, V) grow exponentially as shown in equations [36 a] and [36 b]. The second derivative $\frac{d^2 V}{d V_0^2}(\Theta)$ is positive if $\Theta > \Theta_\infty$. It is negative if $\Theta_\infty > \Theta > \Theta_c$

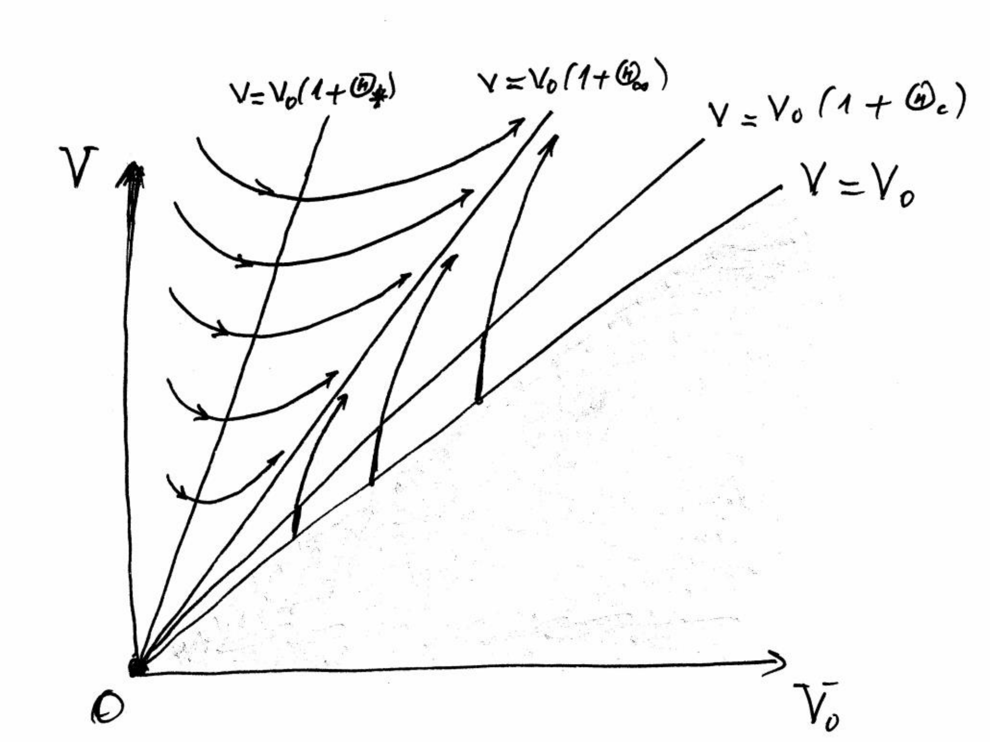

Figure 3. A qualitative sketch of the orbits when $\Delta\pi > \Delta P_c$

As there are two qualitatively different classes of phase portraits, one for $\Delta\pi < \Delta P_c$ and the other for $\Delta\pi > \Delta P_c$**,** the difference in osmotic pressure $\Delta\pi$ is thus a bifurcation parameter, whose critical value is $\Delta\pi = \Delta P_c$**.**

(2.2.2)-The non-autonomous case.

Having justified Lockhart hypothesis as an asymptotic property of the relative rates of variation of the volume of the turgid cell and the cell in incipient plasmolysis for the autonomous case, let us see now if it is possible to justify the

hypothesis in the non-autonomous case, in which the osmotic pressure and the elastoplastic properties of the cell primary wall suffer variations during the growth process.

From now on we assume that $h\left(t, g\left(t,\Theta\right)\right) > 0$**: there is always growth.**

Let us write again equations [19] and [20]:
$$\frac{d\,\Theta}{d\,t} = \left(1+\Theta\right)c\left(t,\Theta\right) = f\left(t,\Theta\right) \qquad [39]$$

$$c\left(t,\Theta\right) = a\left(t,\Theta\right) - b\left(t,\Theta\right) = \mu K_H\,\left(\Delta\pi\left(t\right) - g\left(t,\Theta\right)\right) - h\left(t, g\left(t,\Theta\right)\right) \qquad [40]$$

Now the difference in osmotic pressure $\Delta\pi\left(t\right)$ is not constant: it varies first in the fast in the time scale of plasmolysis when the turgid volume $V\left(t\right)$ relaxes from its initial value $V\left(0\right)$ towards the more slowly varying values it takes during the growth time scale. So, we assume that $\Delta\pi\left(t\right)$ is bounded from above and from below.

The fast relaxation of the turgid volume during plasmolysis, and its relatively slow variation (in comparison with the time scale of plasmolysis) during growth, suggests that the boundary layer approach can be applied to the problem of constructing a solution.

To study the non-autonomous case the singular perturbations approach summarized in the Appendix (5.2) can be applied. The dependent variable z that appears there now is Θ. The positive parameter ε that appears in left hand side of equation [A1] in Appendix (5.2) can be estimated by the quotient $\frac{\tau_i}{\tau_o}$ between the inner time scale $\tau_i = \tau_p$ of plasmolysis and a measure of the outer time scale $\tau_o = \tau_g$ related with the process of growth.

However, this small parameter ε doesn't appear in the equation [39] because the meaning of the time variable is different in [A1] and in [39]. In [A1] the time variable t can be considered as dimensionless. It takes values $O(1)$ when the clock time takes values near $\tau_o = \tau_g$ and takes values near $O\left(\varepsilon\right)$ when the clock time takes values near $\tau_i = \tau_p$.

In [39] the time variable t must be interpreted as clock time.

Following the procedure sketched in the Appendix, we consider first the degenerate equation $f\left(t,\Theta\right) = 0$

This equation has only one positive root $\Theta_\infty\left(t\right)$, and it is globally stable. This is so because when $0 \le \Theta\left(t\right) < \Theta_\infty\left(t\right)$ then $f\left(t,\Theta\left(t\right)\right) > 0$, and when $\Theta\left(t\right) > \Theta_\infty\left(t\right)$ then $f\left(t,\Theta\left(t\right)\right) < 0$.

The other root is $\Theta = -1$, being unphysical can be neglected.

The equation for the inner solution is $\frac{d\,\Theta_i}{d\,t} = f\left(0,\,\Theta_i\right)$ with the initial condition $\Theta_i\left(0\right) = \Theta\left(0\right)$.

The outer solution is now the function $\Theta_\infty\left(t\right)$, so according to equation [A3] of (5.2) we construct the approximate solution:
$$\Theta_{app}\left(t\right) = \Theta_i\left(t\right) + \Theta_\infty\left(t\right) - \Theta_\infty\left(0\right) \qquad [41]$$

To estimate the thickness of the boundary layer, we introduce the new variable $\xi\left(t\right) = \Theta_i\left(t\right) - \Theta_\infty\left(0\right)$ and make a linear approximation of $\frac{d\,\Theta_i}{d\,t} = f\left(0,\,\Theta_i\right)$ in a neighborhood of $\xi = 0$:
$$\frac{d\,\xi}{d\,t} = -\frac{1}{\tau_i}\xi \qquad [42]$$

Considering that $\frac{\partial}{\partial\Theta}c\left(0,\Theta_\infty\left(0\right)\right)$ is negative, the inner time scale is:

$$\tau_i = \frac{1}{\left(1+\Theta_\infty\left(0\right)\right)\left|\frac{\partial}{\partial\Theta}c\left(0,\Theta_\infty\left(0\right)\right)\right|} \qquad [43]$$

The outer time scale $\tau_o = \tau_g$ is the characteristic time of variation of the outer solution $\Theta_\infty\left(t\right)$ in the time interval $\left[0,T\right]$ during which the growth process is studied:

$$\tau_o = \tau_g = \frac{\max\left\{\left|\Theta_\infty(t)\right|\right\}_{t\in[0,T]}}{\max\left\{\left|\frac{d}{dt}\Theta_\infty(t)\right|\right\}_{t\in[0,T]}} \qquad [44]$$

After an initial time interval of, say, extension $3\tau_i$ the inner solution is already relaxed to $\Theta_\infty(0)$ and the approximate solution reduces to the stable root of the degenerate equation $\Theta_{app}(t) = \Theta_\infty(t)$.

Because $c(t,\Theta_\infty(t)) = 0$, we deduce the following equalities (being the last one the definition of $\lambda_\infty(t)$):

$$a(t,\Theta_\infty(t)) = b(t,\Theta_\infty(t)) = \lambda_\infty(t) \qquad [45]$$

Then by a suitable choice of $\bar{V}(0)$ and $\bar{V}_0(0)$ equations [21 a] and [21 b] can be recast as the following **external approximations** to the turgid volume and the volume in incipient plasmolysis:

$$V(t) = \bar{V}(0)\exp\left[\int_0^t \lambda_\infty(s)\,ds\right] \qquad [46\text{ a}] \qquad\qquad V_0(t) = \bar{V}_0(0)\exp\left[\int_0^t \lambda_\infty(s)\,ds\right] \qquad [46\text{ b}]$$

$$\bar{V}(0) = V(0)\exp\left[\int_0^{3\tau_i}\left\{a(s,\Theta_\infty(s)) - \lambda_\infty(s)\right\}ds\right] \qquad [47\text{ a}]$$

$$\bar{V}_0(0) = V_0(0)\exp\left[\int_0^{3\tau_i}\left\{b(s,\Theta_\infty(s)) - \lambda_\infty(s)\right\}ds\right] \qquad [47\text{ b}]$$

From equations [18 a] and [18 b], considering [44], or directly from [45 a] and [45 b] we have asymptotically:

$$\frac{1}{V}\frac{dV}{dt} = \frac{1}{V_0}\frac{dV_0}{dt} = \lambda_\infty(t) \qquad [48]$$

But this is Lockhart's hypothesis extended to the non-autonomous case.
The numerical error made by accepting the Lockhart hypothesis at any time during the process is given by:

$$\left|\frac{1}{V}\frac{dV}{dt} - \frac{1}{V_0}\frac{dV_0}{dt}\right| = \left|c\left(t,\Theta\left(t;\Theta(0)\right)\right)\right| \qquad [49]$$

In [49] appears the exact solution $\Theta\left(t;\Theta(0)\right)$ of equation [39] for the initial condition $\Theta(0)$.

If $\Theta_{app}(t)$ is the approximate solution with the same initial condition, the local values of the numerical error during the process can be estimated by:

$$\left|\frac{1}{V}\frac{dV}{dt} - \frac{1}{V_0}\frac{dV_0}{dt}\right| \simeq \left|c\left(t,\Theta_{app}(t)\right)\right| \qquad [50]$$

Considering the error bounds that can be established for the approximate solution, it is possible to give upper bounds the local values given by [50].

(3)-Discussion and conclusions

(3.1)-Some special cases of elastic and plastic strain of the primary plant cell wall: the spherical osmometer in the autonomous case and the linear osmometer in the non-autonomous case

(3.1.1) Linear elastic spherical osmometer and the possibility of a cell burst after a finite time interval.

Let us first compare the Lockhart's growth equation for a cylindrical cell wall that elongates without variation of its radius, with the case of a spherical cell wall with isotropic elastic and plastic properties that expands radially maintaining its spherical form.

For a spherical cell wall $V=\frac{4}{3}\pi R^3$ and $V_0=\frac{4}{3}\pi R_0^{\ 3}$ while $\mu=\frac{S}{V}=\frac{3}{R}$ and $\frac{1}{V}\frac{dV}{dt}=\frac{3}{R}\frac{dR}{dt}$

So, equation [1 b] reduces to the following one:
$$\frac{dR}{dt}=K_H\left(\Delta\pi-\Delta P\right) \qquad [51]$$

Because the quotient $\frac{\delta}{R}$ between the cell wall thickness δ and the cell radius R is very small relative to 1, assuming that the wall is in mechanical equilibrium, the turgor pressure ΔP can be related with the mean stress σ in the wall by the following formula (Timoshenko and Goodier, 1951):
$$\Delta P=\frac{2\delta}{R}\sigma \qquad [52]$$

The stress-strain constitutive relation for the elastic material of the spherical shell is (Timoshenko and Goodier, 1951):
$$\sigma=E\left(\frac{R-R_0}{R_0}\right) \qquad [53]$$

Here E is a representative value of the Young's module of the primary wall.

From [52] and [53] we obtain:
$$\Delta P=2\ \delta E\left(\frac{1}{R_0}-\frac{1}{R}\right) \qquad [54]$$

From [51] and [54], in the time scale of cell growth in which $\Delta\pi$ can be considered as independent of the wall radius:
$$\frac{dR}{dt}=K_H\left(\Delta\pi+2\ \delta E\left(\frac{1}{R}-\frac{1}{R_0}\right)\right) \qquad [55]$$

If the irreversible extensibility of the wall ϕ_0 is zero, R_0 remains fixed. If $\Delta\pi$ remains fixed, from [] it follows that there is only one equilibrium radius, with physical sense if and only if $\frac{R_0\,\Delta\pi}{2\ \delta E}<1$:
$$R_*=\frac{R_0}{\left(1-\frac{R_0\,\Delta\pi}{2\ \delta E}\right)} \qquad [56]$$

If we introduce, by definition, the function $V(R)=\left(R-R_*\right)^2$, from [55] and [56] we have for every positive cell radius $R\neq R_*$ (being $R_*>0$):
$$\frac{d}{dt}V(R)=\frac{d}{dt}\left(R-R_*\right)^2=-4\ \delta E\,K_H\ \frac{\left(R-R_*\right)^2}{R_*R}<0 \qquad [57]$$

Consequently $V(R)=\left(R-R_*\right)^2$ is a Liapunov's function such that the equilibrium radius is globally and asymptotically stable (Malkin, 1952; Coddington and Levinson, 1955).

However, if a linear elastic behavior of the cell wall material is assumed in a cell that expands in an isotropic way, a loss of stability appears when the difference in osmotic pressure $\Delta\pi$ exceeds a critical value $\Delta\pi_c=\frac{2\delta E}{R_0}$

According to the mathematical model, if the material continues to behave as linear elastic, R_* would be devoid of physical sense (it would be negative). The cell should burst (the radius would tend to $+\infty$) after a finite time inter-

val. This can be seen directly because the differential equation [55] can be integrated by separation of variables. The time instant is obtained as an analytic function of cell radius and the behavior in the large can be studied also and with more detail from this function.

This instability does not appear when the growth is completely anisotropic, as is the case described by Lockhart's axial growth model, in which a linear elastic behavior of the wall is also assumed.
To see this, let us suppose again that the irreversible extensibility of the wall ϕ_0 is zero. Then the length L_0 of the cell in incipient plasmolysis remains constant in equation [13 a]:

$$\frac{1}{L}\frac{dL}{dt} = \frac{2}{R}K_H\left(\Delta\pi - \frac{2\delta}{R}E\left(\frac{L-L_0}{L_0}\right)\right) \qquad \text{[13 a bis]}$$

If $\Delta\pi$ remains fixed, from [13 a bis] it follows that there is only one equilibrium length L_∞ that (is globally asymptotically stable and) remains finite for any finite $\Delta\pi$:

$$\frac{L_\infty}{L_0} = 1 + \frac{R\Delta\pi}{2\,\delta E}$$

(3.1.2)-The equation of the linear osmometer in the non-autonomous case

The equation of the linear osmometer in the non-autonomous case when the plastic yield stress in the wall can be neglected can be written, considering that $\Theta = \dfrac{L-L_0}{L_0}$ $\quad \Delta P = \dfrac{2\delta}{R}\sigma \quad \Delta P_c = 0 \quad \sigma = E\,\Theta \quad h(\Delta P) = \beta_0\,\Delta P$:

$$\frac{d\Theta}{dt} = \left(\frac{2\delta(\beta_0 + \mu K_H)E}{R}\right)(1+\Theta)\left(\frac{\mu K_H R\Delta\pi}{2\delta(\beta_0+\mu K_H)E} - \Theta\right) \qquad \text{[58]}$$

Introducing the time functions $\alpha(t) = \dfrac{2\delta(\beta_0 + \mu K_H)E}{R}$ and $\beta(t) = \dfrac{\mu K_H R\Delta\pi}{2\delta(\beta_0+\mu K_H)E}$ we see that equation [58] is a Riccati's equation with variable coefficients (Davis, 1962):

$$\frac{d\Theta}{dt} = \alpha(t)(1+\Theta)(\beta(t) - \Theta) \qquad \text{[59]}$$

As we know the particular (and unphysical) solution $\Theta = -1$, making the change of variable $\Theta = -1 + \dfrac{1}{\psi}$ the equation can be reduced to a linear differential equation of the first order in ψ, whose analytical solution can be found in known ways for any initial condition $\psi(0) = \dfrac{1}{1+\Theta(0)}$. This solution can be used to study in detail the error made when the exact solution is approximated by the method of matched asymptotic expansions. This will not be done here.

(3.2)- Elasticity, plasticity, volume flow and longitudinal growth of the primary cell wall.

Now let us review the results obtained above for the longitudinal growth of the primary plant cell wall focusing in the validity of Lockhart's hypothesis.

(3.2.1) Elasticity, plasticity, volume flow and longitudinal growth in the autonomous case

(a)-For the autonomous case, from equations [28], considering that $V(t) = \pi R^2 L(t)$ and $V_0(t) = \pi R^2 L_0(t)$:

$$\frac{1}{L}\frac{dL}{dt} = a(\Theta) = \mu K_H \left(\Delta\pi - g(\Theta)\right) \qquad \frac{1}{L_0}\frac{dL_0}{dt} = b(\Theta) = h\left(g(\Theta)\right)$$

In these relations $\Delta P = g(\Theta)$ is the elastic characteristic and $\frac{1}{L_0}\frac{dL_0}{dt} = h(\Delta P)$ is the plastic mechanical-chemical characteristic of the cell primary wall. $\frac{1}{L}\frac{dL}{dt} - \frac{1}{L_0}\frac{dL_0}{dt} = c(\Theta) = a(\Theta) - b(\Theta)$, so $\frac{1}{L}\frac{dL}{dt} = \frac{1}{L_0}\frac{dL_0}{dt}$ when $\Theta = \Theta_\infty$ is a root of $c(\Theta) = 0$. This root always exists and is unique.

(b)-The difference in osmotic pressure $\Delta\pi$ is a bifurcation parameter, whose critical value is $\Delta\pi = \Delta P_c$, being ΔP_c is the critical turgor pressure beyond which the plastic, mechanical-chemical expansion of the cell wall begins.

If $\Delta\pi < \Delta P_c$ then $a(\Theta_\infty) = b(\Theta_\infty)$ is zero so there is no asymptotic growth of the primary wall. The portrait in the phase plane is qualitatively as shown in Figure 2.

If $\Delta\pi > \Delta P_c$, both $a(\Theta_\infty)$ and $b(\Theta_\infty)$ are positive and there is a sustained asymptotic exponential growth. The portrait in the phase plane is qualitatively as shown in Figure 3.

(c)-All possible trajectories of cubic dilatation $\Theta(t;\Theta(0))$ that approach to their rest point Θ_∞ have the same Liapunov-Perron number τ_Θ. This number gives a measure of the time scale of the relaxation of the cubic dilatation:

$$\frac{\Theta(t) - \Theta_\infty}{\Theta(0) - \Theta_\infty} \approx \exp\left[-\frac{t}{\tau_\Theta}\right]$$

(d)- From [36 a] and [36 b], considering that $V(t) = \pi R^2 L(t)$ and $V_0(t) = \pi R^2 L_0(t)$, we have asymptotically when $t \to +\infty$: $L(t) \approx \bar{L}(0)\exp[\lambda_\infty t]$ $\qquad L_0(t) \approx \bar{L}_0(0)\exp[\lambda_\infty t]$ where $\frac{L(0)}{\bar{L}_0(0)} = 1 + \Theta_\infty$

The Liapunov-Perron numbers for the turgid cell length $L(t)$ and the length $L_0(t)$ at incipient plasmolysis are both equal to λ_∞.

(e)-The hypothesis $\frac{1}{L}\frac{dL}{dt} = \frac{1}{L_0}\frac{dL_0}{dt}$ introduced by Lockhart is verified asymptotically when time tends to infinity.

(3.2.2)-Elasticity, plasticity, volume flow and longitudinal growth in the non-autonomous case

(a)-Integrating equation [42] for the linearized relaxation of the inner solution we obtain:

$$\frac{\Theta_i(t) - \Theta_\infty(0)}{\Theta(0) - \Theta_\infty(0)} = \exp\left[-\frac{t}{\tau_i}\right] \qquad [60]$$

From [] we see that after $t > 3\tau_i$ we can put $\Theta_i(t) - \Theta_\infty(0)$ with a good approximation.

After this, $\Theta(t)$ follows very closely the time variation of the stable root of the degenerate equation (the external approximate solution) $\Theta_\infty(t)$.

(b)-From [46 a] and [46 b] we obtain:

$$L(t) \approx \bar{L}(0)\exp\left[\int_0^t \lambda_\infty(s)\,ds\right] \qquad [61\ a]$$

$$L_0(t) \approx \bar{L}_0(0)\exp\left[\int_0^t \lambda_\infty(s)\,ds\right] \qquad [61\ b]$$

(c)-From [48]:

$$\frac{1}{L}\frac{d\,L}{d\,t}=\frac{1}{L_0}\frac{d\,L_0}{d\,t}=\lambda_\infty\left(t\right) \qquad [62]$$

(d)-From [50] the numerical error produced when Lockhart's hypothesis is accepted can be estimated in each instant of time using the approximate solution obtained by the method of matched asymptotic expansions. Establishing upper bounds for $\left|c\left(t,\Theta_{a\,p\,p}\left(t\right)\right)\right|$ allows bound the numerical error associated with Lockhart's hypothesis.

(3. 3)-Comments about the strength and weakness of mathematical modeling

On the scale of growth some of the parameters that were introduced by Lockhart in the mathematical model may undergo non-negligible modifications, in time and sometimes also in space (non-spatial homogeneity).

Thus, in the axial elongation experiments carried out by Bürstrom, the global Young´s modules first increase slightly, then decrease up to 10% of their maximum values and finally increase again until recovering values close to the initial ones (Frey-Wissling, 1952).

The available evidence suggests that variations in mechanical stresses and deformations that are associated with the growth of the primary wall are fed back to several of the cellular systems, influencing the cytoskeleton, the transport of auxins and the cellulose deposit on the inner face of the wall. So, the class of generic mathematical models of cell wall extension discussed in this article, although considering possible time variations in the extensibility, osmotic pressure, water permeability, critical turgor pressure and elasticity parameters of the wall vary during growth, they are, however, very crude approaches that leave in the shade what happens in relation to the mechanical-chemical interactions in the wall of a plant cell.

Since the fifties, within the framework afforded by the central dogma of molecular biology, research in the molecular aspects of biological process has been increasing faster and faster. This can be seen in the contents of exhaustive textbooks of biochemistry (Lehninger, 1975) and is making its way into relatively conservative disciplines such as plant physiology (Noble, 1974) and medical physiology (Meyer, 1983).

In molecular biology, mathematical models have not been used significantly since the type of research that is carried out does not require it. Furthermore, the wealth of details is overwhelming, so it is impossible to take care of all of them in a mathematical model, as some biologist would like.

However, if mathematical modeling is done with enough biological knowledge and from a well-defined and suitably selected point of view, the process of building a mathematical model makes the researcher concentrate on separating the essential from the non-essential according to the chosen perspective for the research. (In fact, this simplification happens inevitably when building any model, even a verbal one.)

When it is well conceived and constructed, a simple mathematical model can reveal some essential elements that make up a complex phenomenon.

Being stripped of details that in the first instance can be considered secondary, it is expected that a simple model will allow us to deal more easily with the analysis of certain interactions that, when working with a more complicated model structure, either mathematical or verbal, could remain hidden.

As the mathematician Mark Kac said once, the models are cartoons of reality, but if they are good, like good cartoons, they portray, albeit in a distorted way, some features of the real world. They are fun to invent and to play, and they have a particular life that is their own.
"But what gives these models a hold on life – tenuous though this hold may be – is that despite admitted lack of realism, they are firmly rooted, and they were conceived to deal with real questions. Without such rooting and without

real questions to guide us, we may well find ourselves fighting windmills and triumphantly emerging with pyrrhic victories.” (Kac, 1972)

Indeed, that kind of own life that has a mathematical model is both its greatest strength and its greatest weakness. If it is good, it broadens the field of vision of the researcher, suggests new experiments that without their help could not have been conceived, faithfully represents some essential aspects of the real system, and lends itself to be improved in successive stages of adjustment until reaching a sufficient precision to be used in practice both in medicine and in technology or in research in pure science. But if it is bad, its blinding effects are more intense than those of a bad verbal model: it not only deforms reality beyond the permissible but can lead to questions without real meaning and to the design of experiments completely divorced from the real system that the researcher intends to investigate.

(4)-Acknowledgements

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(5)-Appendix

(5.1) Auxiliary lemma

Let us suppose that the real function $f(t)$ is defined in $[0,+\infty)$ and is absolutely integrable in every $[0,t]$.

Then, if $\lim_{t\to\infty} f(t)=\alpha$ it follows that $\lim_{t\to\infty}\left(\frac{1}{t}\int_0^t f(t)\,dt\right)=\alpha$

Effectively from the existence of the limit, for every $\varepsilon>0$ there must exist one $t_0(\varepsilon)>0$ such that if $t>t_0(\varepsilon)$, then $|f(t)-\alpha|<\varepsilon$.

If $t>t_0(\varepsilon)$: $\left|\frac{1}{t}\int_0^t (f(s)-\alpha)\,ds\right| \le \left|\frac{1}{t}\int_0^{t_0(\varepsilon)} (f(s)-\alpha)\,ds\right| + \left|\frac{1}{t}\int_{t_0(\varepsilon)}^{t} (f(s)-\alpha)\,ds\right| \le \left|\frac{1}{t}\int_0^{t_0(\varepsilon)} (f(s)-\alpha)\,ds\right| + \varepsilon$

Taking the limit for $t\to+\infty$ we obtain $\lim_{t\to\infty}\frac{1}{t}\int_0^t (f(s)-\alpha)\,ds=0$

From this last result and from the equality $\frac{1}{t}\int_0^t f(s)\,ds-\alpha=\frac{1}{t}\int_0^t (f(s)-\alpha)\,ds$ we obtain $\lim_{t\to\infty}\left(\frac{1}{t}\int_0^t f(t)\,dt\right)=\alpha$

(5.2) Singular perturbations, boundary layers, inner solutions, outer solutions, and slow manifolds

Singular perturbation methods can be classified in two classes: the so called “layer problems” that are approached by the method of matched asymptotic expansions and the multiple scale problems that are approached by the method of multiple scales (Bender and Orszag, 1978).

Finding an approximate solution for the non-autonomous system of nonlinear differential equations that describes plasmolysis processes and growth of the primary cell wall requires a boundary layer approach.

The boundary layer, in this case, is an interval of time beginning at $t=0$ and with an extension of the numerical order of the time scale of the plasmolysis. In this narrow region of time (narrow relative to the duration of the wall growth) the turgid volume can change very fast in relation with its variation during the growth process itself.

To summarize the method of matched asymptotic expansions, let us consider the following differential equation, with a small positive parameter ε (small relative to 1), and initial condition $z(0)=z^0$:

$$\varepsilon \frac{d}{dt} z = f(t, z) \qquad \text{[A1]}$$

We assume that the function $f(t,z)$ is regular enough for $0 \le t \le T$ and $z \in I$ being I an interval of values of z big enough to include the initial points of interest.
If we put $\varepsilon = 0$, from [A1] we obtain the so-called degenerate equation:

$$f(t,z) = 0 \qquad \text{[A2]}$$

Let us suppose that the degenerate equation has three roots $z = \varphi_1(t)$, $z = \varphi_2(t)$ and $z = \varphi_3(t)$ so $f(t, \varphi_j(t)) = 0$ for every $t \in [0,T]$, $j \in \{1,2,3\}$.

Besides, let us suppose that $f(t,z)$ changes it sign at every transition through a root, as shown in Figure 4:

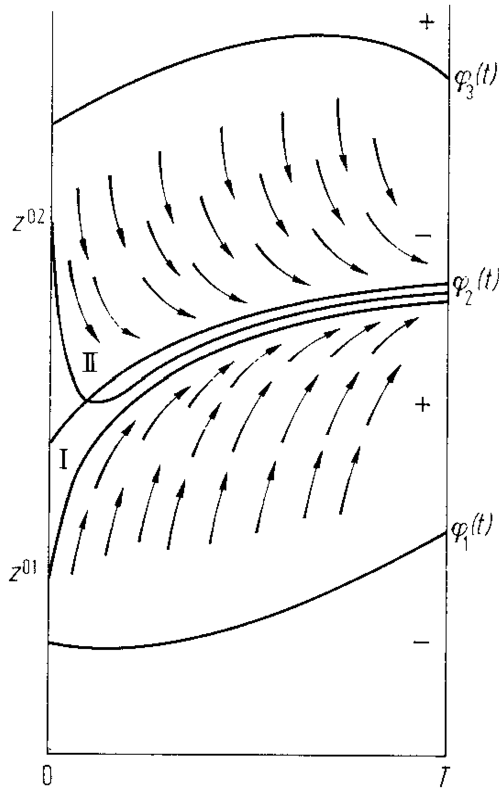

Figure 3. A sketch of the three roots of the degenerate equation $f(t,z) = 0$ and its relations with the field of directions of $\varepsilon \frac{d}{dt} z = f(t,z)$ and with two particular solutions of this equation with initial conditions $z(0) = z^{02}$ and $z(0) = z^{01}$
(Taken from Mathematics Applied to Physics, E. Roubine (Editor), UNESCO Press, Paris, France, 1970.)

A solution of [A1] that crosses one of the functions $\varphi_j(t)$, does it horizontally, as show Figure 4 for the solution with initial value $z(0) = z^{02}$. This can happen because $\varphi_j(t)$ are not solutions of the original differential equation.
In general the initial value of the roots $\varphi_j(0) \neq z^0 = z(0)$ However, the root $z = \varphi_2(t)$ is stable, attracts the trajectories that begin in points located between $z = \varphi_1(t)$ and $z = \varphi_3(t)$, while these other two roots act like repelling

these trajectories (are unstable). So, the stable root $z=\varphi_2(t)$ has a domain of influence bounded by the other two roots, such that after a short time interval (a boundary layer whose extension is measured by the positive parameter ε) the trajectories contained between these two boundaries approach fast to $z=\varphi_2(t)$ and remain in its neighborhood, now varying with the slow time scale of the function $\varphi_2(t)$. As time increases, the solutions $z(t;\, z^0)$ of the differential equation remain closer to the solution $z=\varphi_2(t)$ of the degenerate equation, the smaller is ε.

The inequality $\left[\dfrac{\partial}{\partial z} f(t,z)\right]_{z=\varphi(t)} < 0$ for every $t\in[0,T]$, affords an analytical indication of stability for the solution $\varphi(t)$ of the degenerate equation.

A generalization of this kind of approach to singular perturbation problems and its mathematical foundation, can be found in the contents and bibliography of a chapter written by Tihonov, Vasileva and Volosov (Tihonov et al, 1970).

In the general case of a system of coupled differential equations, some dependent variables present fast time variations and are enslaved by the other slower variables. Assuming that the fast variables are relaxed to equilibrium with slow variables, a system of degenerate equations is obtained. Between the solutions of the degenerate equations, we find the **stable slow manifolds**. These manifolds attract to their neighborhood the orbits that begin in their attraction basins. As already said in note 4, a slow manifold taken in this sense, is not an invariant manifold, because it is not made with some of the orbits of the dynamical system.

An approximate solution for the initial value problem $\varepsilon\dfrac{d}{dt}z = f(t,z),\, z(0)=z^0$ can be constructed combining two solutions, the inner and the outer one (Lin and Segel, 1974).

The **inner solution** $z_i(t)$ solves the initial value problem $\varepsilon\dfrac{d}{dt}z = f(0,z),\, z(0)=z^0\in\left(\varphi_1(0),\varphi_3(0)\right)$ and the **outer solution** $z_o(t)$ is the stable root $z=\varphi_2(t)$.

We expect that the inner solution approximates the exact solution in a short time interval (an internal layer of thickness that decreases with the positive parameter ε) adjacent to the initial instant $t=0$, and the outer solution approximates the exact solution in the rest of the interval $[0,\, T]$.

In the transition region between the inner boundary layer and the nearest (to the boundary layer) part of the rest of the interval $[0,\, T]$, both solutions should be good approximations to the exact solution when ε is small.

Considering all this the following combination of the inner and the outer solution can be used as an approximation to the exact solution:

$$z_{app}(t) = z_i(t) + z_o(t) - z_o(0) = z_i(t) + \varphi_2(t) - \varphi_2(0) \qquad [A3]$$

The inner solution $z_i(t)$ satisfies the initial value condition, so the same happens with $z_{app}(t)$:

$$z_{app}(0) = z_i(0) + \varphi_2(0) - \varphi_2(0) = z^0$$

Being a solution of $\varepsilon\dfrac{d}{dt}z = f(0,z)$, $z_i(t)$ tends to $\varphi_2(0)$, which is the only equilibrium point of this differential equation when $z^0\in\left(\varphi_1(0),\varphi_3(0)\right)$. The rest point $\varphi_2(0)$ attracts all the solutions whose initial values are in $\left(\varphi_1(0),\varphi_3(0)\right)$.

As will be shown now, **once** $f(t,z)$ **becomes small, it remains small.**

To see this, let us consider the following **Theorem**:

Let us extend the interval $[0,T]$ *to* $[0,+\infty)$ *and suppose that both* $f(t,z)$ *and* $\frac{\partial}{\partial t} f(t,z)$ *are bounded and* $\frac{\partial}{\partial z} f(t,z) < -k < 0$ *for all* t, z *in the domain of influence of a stable root of the degenerate equation.*

If for some instant $t = t_0$ *we have* $\left[f(t,z) \right]^2 < \varepsilon$, *then* $\left[f(t,z) \right]^2 < \varepsilon$ *for all* $t \geq t_0$ *and* ε *small enough, being* ε *the parameter that appears in the left hand side of equation* [A1].

If the theorem is not true, there will be an instant $t = t_*$ such that $\left[f(t_*, z) \right]^2 = \varepsilon$ and $\frac{d}{dt}\left[f(t_*, z) \right]^2 \geq 0$.

However, $\frac{d}{dt}\left[f(t,z) \right]^2 = 2 f \left(\frac{\partial}{\partial t} f(t,z) + \frac{\partial}{\partial z} f(t,z) \frac{d}{dt} z \right) = 2 \left(f \frac{\partial}{\partial t} f(t,z) + \frac{f^2}{\varepsilon} \frac{\partial}{\partial z} f(t,z) \right)$

Considering that $f^2(t*, z) = \varepsilon$: $\quad f(t*, z) \frac{\partial}{\partial t} f(t_*, z) + \frac{f^2(t*, z)}{\varepsilon} \frac{\partial}{\partial z} f(t*, z) \leq \sqrt{\varepsilon} \frac{\partial}{\partial t} f(t*, z) - k$

So for ε small enough $\frac{d}{dt}\left[f(t_*, z) \right]^2 < 0$, which contradicts the hypothesis $\frac{d}{dt}\left[f(t_*, z) \right]^2 \geq 0$, so the theorem must be true, and $\sqrt{\varepsilon}$ gives a measure of $\left| f(t,z) \right|$ after the solution leaves the inner region (the boundary layer).

(6)-References

Coddington E. and Levinson N., Theory of ordinary differential equations, Mc Graw-Hill, New York, 1955.

Bender, C. M. and Orszag S. A., Advanced mathematical methods for scientists and engineers I: asymptotic methods and perturbation theory, Mc Graw-Hill, New York, 1978.

Dainty, J., Water relations of plant cells, Advances in Botanical Research, 1963, **1**:279-326.

Davis, H. T., Introduction to nonlinear differential and integral equations, Dover, New York, 1962.

Erickson, R.O., Microfibrillar structure of growing plant cell walls En Getz, W. M. (ed) Mathematical Modeling in Biology and Ecology, Lecture Notes in Biomathematics, vol. 33, Springer-Verlag, Berlin, 1980.

Frey-Wyssling A., Deformation of Plant Cell Walls, in Frey-Wyssling A. (Editor) Deformation and Flow in Biological Systems, Interscience, New York, 1952, 194-254.

Green P., Erickson R. and Buggy J., Metabolic and physical control of cell elongation rate, Plant Physiology, 1971, **47**:423-430.

Han, W., Stability of motion, Springer-Verlag, Berlin, 1967.

Haken H., Advanced Synergetics, Springer-Verlag, Berlin, 1983.

Harris, E., Transport and accumulation in biological systems, Butterworth, London, 1960.

Hirsch, M. and Smale, S., Differential equations, dynamic systems and linear algebra, Academic Press, New York, 1974.

Kac, M., On Applying Mathematics: Reflections and Examples, Qt. Appl. Math. 1972, **30**: 17–29

Katchalsky A. and Curran F., Nonequilibrium Thermodynamics in Biophysics, Harvard University Press, Cambridge MA, 1967.

Lehninger, A.L., Biochemistry, 2nd Edition, Worth, New York, 1975.

Liapunov, A. M., Problème Général de la Stabilité du Mouvement, Annals of Mathematical Studies Nº 17, Princeton University Press, Princeton, New Jersey, 1947 (Liapunov's original research article was published in 1907).

Lin C. and Segel L., Mathematics Applied to Deterministic Problems in the Natural Sciences, Mc. Millan, New York, 1974.

Lockhart J. A., An analysis of irreversible plant cell elongation, Journal of Theoretical Biology 1965, **8**:264-275.

Malkin, I. G., Theory of the stability of motion, US Atomic Energy Commission, Office of Technical Information, New York, 1952.

Meyer, Ph. (Editor), Physiologie humaine, Flammarion, Paris, 2nd Edition, 1983.

Mühlethaler, K., Ultrastructure and formation of plant cell walls, Annual Reviews of Physiology, 1967, **18**:1-24.

Noble P., Biophysical Plant Physiology, Freeman, San Francisco, 1974.

Perron, O., Die Ordnungszahlen linearer Differentialgleichungssysteme, Math. Zeits. 1930, **31**:748-766.

Saaty, Th. L. and Bram J., Nonlinear Mathematics, Dover, New York, 1981.

Suárez-Antola, R. Elasticidad, Plasticidad y Flujo de Volumen en las Células Vegetales (Elasticity, plasticity and volume flow in plant cells), book to be published in 1985. **Now available through researchgate**.

Tihonov A., Vasileva A. and Volosov V., Ordinary differential equations, in Mathematics Applied to Physics, E. Roubine (Editor), UNESCO Press, Paris, France,1970.

Timoshenko, S. and Goodier J. H., Theory of Elasticity, Mc-Graw Hill, New York, 1951.

Wainwright S., Biggs W., Currey J. and Gosline J., Mechanical Design in Organisms, Princeton University Press, Princeton, New Jersey, 1976.